NAVAL SUBMARINE MEDICAL RESEARCH LABORATORY

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HYPERBARIC AND HYPEROXIC EFFECTS ON
PULMONARY FUNCTION DURING AIR SATURATION DIVES

J. H. Dougherty, JR., R. G. Eckenhoff,[1] W. L. Hunter, JR.,[2] J. W. Parker,
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Naval Medical Research and Development Command
Research Work Unit 63706 M0099PN01C-0007

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SUMMARY PAGE

PROBLEM

Compressed air is a commonly used breathing media in a wide variety of situations. These include undersea diving, pressurized construction, hyperbaric therapy, and certain emergency situations such as sunken vessels or distressed submersibles. Compressed air contains an elevated partial pressure of oxygen (O_2), and since many of these exposures may be prolonged those exposed are at risk of developing pulmonary oxygen toxicity (POT). The purpose of this project was to determine the biological/medical effects of a possible submarine rescue procedure developed for the Deep Submergence Rescue Vehicle (DSRV). The DSRV system has no provision for helium-oxygen diving. Most of the previous studies have utilized high levels of O_2 for relatively short time periods. The present study utilized a large number (35) of subjects exposed to a relatively low-level hyperoxia for extended periods.

FINDINGS

These saturation dive profiles (11 dives) utilizing a total of 35 human subjects were completed. There was an increase of POT as evidenced by signs and symptoms and by forced vital capacity (FVC) and pulmonary diffusing capacity decrements as we progressed from AIRSAT-1 and 2 to 3 for these long-term exposures (dry chamber dives) with relatively low levels of hyperoxia. We found a large individual variation in susceptibility to hyperoxia. All subjects exposed to these levels of inspired O_2 eventually returned to their own predive FVC after O_2 levels were returned to normal. This required from two days to two weeks and occasionally longer.

APPLICATION

Humans can tolerate hyperbaric air at 2.8 ATA (P_{O_2} = 0.60 ATA) with brief exposure to even higher levels for periods at least a week with only minimal signs and symptoms consistent with pulmoanry oxygen toxicity. They can tolerate 24 hours exposure to 5 ATA air (P_{O_2} = 800 torr) with a subsequent air decompression, and demonstrate only moderate symptoms and mild to moderate decrements in the vital capacity. The operational rescue scenarios of the DSRV concept can thus be accomplished for the O_2 levels and durations tested without permanent harm to the rescuees.

ADMINISTRATIVE INFORMATION

This investigation was conducted as part of Naval Medical Research and Devlopment Command Research Work Unit M0099PN01C-0007 — "Evauation of the contribution of myoneural toxicity to decrements in ventilatory indices of pulmonary oxygen toxicity during diving". The present report was submitted for review on 2 July 1985, approved for publication on 19 July 1985 and has been designated as NavSubMedRschLab Report No. 1049.

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ABSTRACT

Three saturation dive profiles (11 separate dives) utilizing a total of 35 human subjects were completed. They were AIRSAT-1 (2.82 ATA air saturation, five 8-hour excursions to 4.03 ATA), AIRSAT-2 (2.82 ATA air saturation, six 2-hour excursions to 5.55 ATA), and AIRSAT-3 (0.3 ATA O_2 at 5 ATA, three 5-hour air excursions to 7 ATA). AIRSAT-3 involved a 24 hour exposure to 5 ATA air prior to decompression. Pulmonary function was measured in all subjects throughout the exposures. Signs and symptoms of pulmonary oxygen toxicity and significant decreases in forced vital capacity and single breath diffusing capacity were observed. Marked individual variability was apparent in all exposures. Air at 2.82 ATA (60 fswg, 0.59 ATA O_2) is very close to the oxygen toxicity threshold, and air at 5.00 ATA (132 fswg, 1.05 ATA O_2) is well beyond it.

INTRODUCTION

Compressed air is a commonly used breathing media in a wide variety of situations. These include: undersea diving, pressurized construction, hyperbaric therapy and certain emergency situations such as the rescue of sunken vessels or distressed submersibles(1, 2). Compressed air contains an elevated partial pressure of oxygen, and since many of these exposures may be prolonged, those exposed are at risk of developing pulmonary oxygen toxicity (POT). A few reports in the literature describe signs and symptoms of POT in humans exposed to compressed air at as little as 2.8 ATA air pressure for periods of time in excess of a week (3), and as great as 5 ATA pressure for 48 hours (4). Because of a unique situation in U.S. Navy submarine rescue capabilities, humans might be unavoidably exposed to compressed air for periods in excess of 48 hours. In addition, changes in pressure (excursions) may occur during the rescue and recovery process. This has the potential for changing the pulmonary oxygen tolerance. We therefore undertook a study of the pulmonary effects of such an exposure as part of a larger, multidisciplinary experiment. This report describes the development of symptoms and pulmonary function changes consistent with POT in a large number of human subjects exposed to compressed air for a prolonged period of time at a variety of pressures.

MATERIALS AND METHODS

Subjects

The subjects were volunteer reserve or active duty military divers with various degrees of diving experience. All had been in hyperbaric chambers previously, but none had been exposed to pressure for at least two weeks preceeding the experiments. Subjects were evaluated predive by an extensive array of physical, pulmonary, laboratory and radiographic studies. None of the subjects had a history of, or findings consistent with, cardiopulmonary disease. The majority of the subjects were athletic, and all were in excellent health. An informed consent was obtained for all subjects prior to any exposures or procedures after having been briefed fully on the purpose, procedures, and possible hazards of the study. The entire experimental protocol for the Air Saturation(AIRSAT) dive series was approved by the Committee for the Protection of Human Subjects at the Naval Submarine Medical Research Laboratory (NSMRL), Groton, Connecticut.

Individual physical characteristics (age, height, weight, and % body fat) as shown in Table 1 were obtained predive. Percent body fat was calculated from skinfold thicknesses (5). A questionnaire on smoking history was completed predive.

Dive Profiles

Three dry saturation dive profiles were accomplished in a large double lock hyperbaric chamber located at NSMRL. The pressurization profiles

(shown in Figure 1) were AIRSAT-1(for Air Saturation-1) (2.82 ATA air saturation, 5 daily 8-hour excursions to 4.03 ATA, 11 subjects), AIRSAT-2 (2.82 ATA air saturation, 6 daily 2-hour excursions to 5.55 ATA, 12 subjects), and AIRSAT-3 (0.3 ATA O_2 at 5.00 ATA saturation, three 5-hour air excursions to 7.00 ATA, 12 subjects). In the latter, there was a shift to air at 5.00 ATA 24 hours before the decompression (breathing air) commenced. The O_2 partial pressure profile for each dive is shown in Figure 2. There were 3 separate AIRSAT-1 dives (3, 4, and 4 subjects), 4 AIRSAT-2 dives (3 subjects each), and 4 AIRSAT-3 dives (3 subjects each). In addition, a control "dive" was performed (CONTROL-1) in which 3 subjects resided in the chamber at 1.06 ATA. They observed the same schedule and performed the same tests as the AIRSAT-3 subjects. The O_2 partial pressure remained essentially normal (0.22) ATA for this exposure.

Testing

Pulmonary monitoring was carried out with a "Wedge" spirometer system, consisting of a Med-Science Electronics Model 370 spirometer and power supply-amplifier unit, and a Model 580 Pulmo Digitizer unit. A Tektronix Model 503 oscilloscope, connected in parallel with the Med-Science equipment, allowed the investigator to monitor and coax the subject. The subjects were standing during the measurements. The spirometer wedge or bellows was inside the chamber, the remaining equipment outside. More detail is given in the third reference (3). This data was collected 1-4 times/day, pre, during, and postdive. During the actual dive period, this test was run at approximately 0900 and 1730 hours for AIRSAT-1 and 2 and 0800 and 1730 hours for AIRSAT-3. It was sometimes run at 2130 hours and occasionally other times if requested by the medical officer for medical monitoring purposes. The excursion dives were from 1000-1800 hours during AIRSAT-1, 1000-1200 hours during AIRSAT-2, and 0900-1400 during AIRSAT-3. The pre and postdive testing was less frequent.

Five parameters were obtained at each sampling period: (1) forced vital capacity (FVC), in liters; (2) forced expiratory volume in 1 second (FEV_1), in liters; (3) forced expiratory volume in 2 seconds (FEV_2), in liters; (4) forced expiratory flow (FEF), in liters/second; and (5) peak inspiratory flow rate (PIFR), in liters/second.

All tests were run in duplicate and the higher of the two values was used. If a value was believed to indicate submaximal effort, an additional maneuver was performed. In most instances, tests were run before meals and after subjects had been upright for some time. Virtually all measurements were made by the same investigator (JHD).

The effect of these exposures on maximal respiratory flow rates (FEF, PIFR, $FEV_{1,2}$/FVC) was analyzed using a method to factor out the well known effects of increased gas density on maximum respiratory flow rates. The use of a formula to convert values at various increased atmosphere pressures to the value they presumably would have been without the decrement caused by the increased gas density. A regression equation ($r = 0.972$) was developed based on unpublished data, from 68 subjects and 109 data pressure points from other experiments (SUREX 1-8 and AIRSAT-1A through 4C) at this

Laboratory. The predive control mean was compared with the earliest compression value for the same man at 2.36, 2.67, 2.82, 2.97, 4.03, and 5.00 ATA. The equation is $y = (RGD)^{-0.416}$ when RGD is the relative gas density of the breathing gas in relationship to air at 1.00 ATA. Then y is multiplied by the actual FEF or PIFR. This is similar to a method used by Wood and Bryan (6, 7) and others (8).

In addition to the wedge data, single breath diffusing capacity was run on at least two different days pre and postdive with a Cardio-Pulmonary Instruments, Inc. Model 5300 Pulmo-Lab. Also measured during AIRSAT 3 (9 subjects only) were the maximum inspiratory and expiratory pressures by means of a U-tube manometer at 3 lung volumes (TLC, FRC, and RV) both pre and postdive. This was to assess ventilatory muscle strength.

Reported signs and symptoms were recorded in permanent logs. Attempts were made not to lead a subject describing his symptoms. Observations of physicians and investigators were routinely recorded. This approach may have resulted in an underreporting of signs and symptoms. In an attempt to estimate the severity of signs and symptoms, a scale with 5 grades was developed: none (0), minimal (+), mild (++), moderate (+++), and severe (++++), based on all available data. Grading of each subjects' signs and symptoms was done by a staff physiologist not familar with the subjects or their pulmonary function results.

Additional data collected included a) hand grip strength from a Smeley type hand grip dynamometer graduated in kilograms (KG), and b) respiratory rate and pulse rate. Subjects were thoroughly trained for all tasks early in the predive period.

Statistics

The values for the individual subjects were pooled for each parameter to obtain a single value for each sampling session. Changes with respect to time were detected by a repeated measures analysis of variance, and the significance of individual points was determined using Dunnett's Test of multiple comparisons. Student's paired t-test was used only when comparing pre and post-exposure data.

RESULTS

Signs and Symptoms

The observed signs and symptoms included trachebronchial irritation, cough, chest tightness, substernal burning, dyspnea, pain during deep breathing (especially during inspiration), sputum production, nausea, and vomiting. Figure 3 shows the time course of signs/symptoms of POT. The percent of the total observed/reported for each day of the dive, decompression, and postdive periods are shown for the 3 dive profiles. Figure 4 shows the number of subjects that were assigned to the different intensities of signs and symptoms. The intensity of signs and symptoms increased and the

FVC decreased (Figure 9) as we proceeded from CONTROL-1 to AIRSAT-1 and 2 to AIRSAT-3 as did the severity of oxygen exposure (Figures 1 and 2). The control subjects had neither signs nor symptoms. There appeared to be a very general relationship between FVC decrements and signs and symptoms, however, no statistically significant relationship could be detected.

Forced Vital Capacity

Figure 5 shows the mean FVC as a percentage of the predive control value (a mean of 4-6 predive values for each subject) for AIRSAT-1 and 2. It can be seen that there was some decrease from the mean control value. There was a substantial individual variation from this slight overall decrement. Examples of the magnitude of individual variation are illustrated in Figures 6 and 7, both from the AIRSAT-3 exposures. Figure 6 represents a resistant subject and Figure 7 shows a more sensitive subject. Despite the large changes, the latter subject recovered quickly. The mean FVC data for AIRSAT-3 (Figure 8) shows a much greater decrement from control than during AIRSAT-1 and 2. There was a 7 day period that the majority of the mean values during the decompression and postdive periods were significantly ($p < 0.05$) depressed.

Figure 9 shows the number of subjects for each exposure that had various decrements in FVC. Because of the motivational nature of the FVC test, a mean of the 3 lowest values is shown to eliminate unwarranted influence by a single spurious point. The other part of this figure shows the number of subjects with various percentages of all post-control sampling periods that were greater than 2 standard deviations below the control value. The shift from minimal to more substantial decrements in FVC as we progressed from CONTROL-1 to AIRSAT-1 and 2 to 3 can be seen by use of either method of presentation.

During the CONTROL-1 Dive (3 subjects, 1.06 ATA) with a normal O_2 tension, the slight changes that occurred in any pulmonary function value were generally opposite in direction (improvements) compared to the other dives (AIRSAT-1, 2, 3). There were never any significant decrements.

All AIRSAT subjects eventually recovered to their own predive control FVC level. There was considerable variation in the time course of recovery. For some subjects this occurred during the dive or immediately postdive. Others required several days to three weeks after the dive for full recovery.

AIRSAT-1 and 2 exposures were associated with few significant changes in respiratory flow rates. AIRSAT-3, on the other hand, showed a significant decrement in FEF and PIFR, coinciding with the FVC changes (Figure 8).

There were no significant changes for the 3 CONTROL-1 Dive subjects for these parameters. Any trends were very slight improvements, presumably due to increases in muscle strength and/or to training.

Diffusing capacity

Due to frequent equipment problems pre and post exposure, data was obtained on 5 subjects for AIRSAT-1, 3 subjects for AIRSAT-2, and 6 subjects for AIRSAT-3 as shown in Table 2. In all cases, the postdive values were lower than the predive values. In AIRSAT-3 these values appeared to decline through the third postdive day, before showing a recovery trend. There was, however, no significant difference between the postdive values. The decreases were statistically significant on postdive days 1, 2, and 3. The other decreases were not significant, possibly because of the small sample size.

Maximum respiratory pressure

Nine subjects in AIRSAT-3 (no data for AIRSAT-1, 2 and 3A) had a decrease in maximum expiratory pressure of 22 mmHg at TLC and 15 mmHg at FRC between mean predive and mean postdive values. The mean maximum inspiratory pressure was decreased 14 mmHg at both FRC and RV. On the other hand, the 3 CONTROL-1 subjects had increases from pre to postdive in all cases (30 mmHg (FRC) and 20 mmHg (RV) for maximum inspiratory pressure and 11 mmHg (TLC) and 5 mmHg (FRC) for maximum expiratory pressure).

Hand grip strength

There was a small decrease in mean hand grip strength from a mean control value of 71.1 to 64.6 KG ($p<.05$) for the 12 AIRSAT-3 subjects (Figure 8). The decrement was observed primarily during the late dive and decompression phase.

Smoking history, age

No relationship between smoking history or age with signs and symptoms or pulmonary function could be established.

DISCUSSION

Signs and Symptoms

The symptoms of cough, chest pain, and tracheobronchitis are similar to those reported by Comroe (9) and Clark and Lambertsen (10,11) with POT. We observed coughing fairly frequently during or immediately following a pulmonary testing maneuver. A few subjects could not hold their breath at TLC during a FVC maneuver when they were experiencing POT because of coughing.

The majority of the signs and symptoms for AIRSAT 1 appeared on the second, third, and seventh days of the dive, (following the first excursion). Most of the AIRSAT 2 signs and symptoms occurred on the second day. Although no direct evidence exists here to implicate an adaptive response to hyperoxia, this would explain the decrease in symptoms and normalization of FVC without a reduction in the inspired PO_2 both AIRSAT-1 and 2. During AIRSAT

3, most of the signs and symptoms occurred after the decompression started; there was a shift to air (1.05 ATA O_2) 24 hours prior to decompression. The incidence for symptoms in AIRSAT-3 was 5 times greater than for AIRSAT-1 and 2. The latency for signs/symptoms to appear and disappear is large enough and variable enough to preclude establishing a relationship with each individual excursion from the saturation or habitat depth.

The signs and symptoms of POT (Fig. 4) were much more apparent during AIRSAT-3 compared to AIRSAT-1 and 2 than the slightly increased oxygen levels from AIRSAT 1 to 2 to 3 as indicated by the Unit Pulmonary Toxic Dose (UPTD) concept of Clark and Lambertsen (10,11) and Wright (12) would account for. This is likely related to the longer time periods at the highest P_{O_2} levels (Figs. 1 and 2). There is less time for the protective effect resulting from intermittent periods of lowered P_{O_2} as reported by Hall (13). Although a general trend for greater symptomatology in the subjects with greater FVC decrements was noted, no support for this relationship was apparent from analysis of the data.

Forced vital capacity

The use of FVC as an indication of POT has been criticized, principally because it is considered not a specific enough test and is subject to motivational factors (2, 14). A recent workshop(2) addressing this problem found little that had a clearcut advantage over the FVC in terms of simplicity and applicability in a hyperbaric environment for monitoring POT in otherwise healthy subjects. In addition there is no experimental support for a more sensitive indicator of POT than the FVC at the present time.

The mechanism(s) of the FVC decrement in POT is still unknown but the following have all been suggested: 1) tissue edema, 2) pulmonary congestion, 3) neuromuscular effects, 4) pain, 5) lack of motivation, 6) hemorrhage, 7) hyaline membrane formation, and others. Many of these factors are only seen in very severe POT, and include chest x-ray changes and gross effects on gas exchange. Our subjects had early, reversible POT, and thus factors 1 and 3-5 are probably more important contributors to the observed FVC decrement. It is likely that the mechanism is multifactorial.

The mean FVC values during all three dive profiles (Figs. 5 and 8) showed an initial decrement after pressurization (days 1 and 2 for AIRSAT-1 and 2 and day 1 for AIRSAT-3) and then a gradual recovery during the following days despite an increasing duration of hyperbaric and hyperoxic exposure. It occurred in the majority, though not all of the individual plots such as are shown in Figs. 6 and 7. This tendency was stronger in individuals that later had the larger FVC decreases. This initial decrease was never statistically significant, however. We have no firm explanation for this observation. It might have been due to the initial stresses (hyperbaric and/or hyperoxic) on the divers. The recovery could also possibly represent a partial adaptation to the hyperoxia.

The 3 CONTROL-1 divers had only one FVC value 2 SD below control; well within the 5% probability. Their mean FVC actually increased slightly during the study. This has been reported to occur with the unaffected sub-

jects in other studies (3, 15-20). One could therefore argue that no increase is actually a slight decrement. Thus the decreases seen in the affected divers from control to the dive and postdive periods may be slightly greater than they appear initially. These increases in the FVC of unaffected divers could be a result of 1) muscle strengthening due to breathing dense gas and/or repeated wedge spirometer testing, and 2) possibly a learning effect.

Because of the slight decrement in FVC (especially when compared to control), the presence of suggestive signs and symptoms, the possible "adaptive" changes, and the highly variable individual response, we suggest that the P_{IO_2} of 0.59 (AIRSAT-1 and 2) lies within the "threshold" range of toxic P_{IO_2}. This agrees with other experiments which show a F_{IO_2} of 60% at 1 ATA is a threshold level (10, 21, 22). AIRSAT-3, on the other hand, showed a more rapid and significant drop in FVC and sign and symptom appearance affirming that a P_{IO_2} of 1.05 is indeed well up the dose-response curve which agrees with clinical experience. The excursions from the saturation depths resulted in brief PIO_2 levels of 0.84, 1.16, and 1.47 ATA for AIRSAT-1, 2, and 3 respectively, and the effect of this on FVC and overall tolerance in our subjects is unknown.

There is a difference in effect on FVC between low level, long-term exposures and higher concentrations experienced for a shorter time period. UPTD values for these exposures are nearly double the 2,100 units that this concept (11,12, 23) predicts would cause a 20% decrement in VC. However, the variation in depths and duration of excursions and the time between excursions which allows for partial recovery significantly complicates these data. The UPTD relationship does not consider these variables.

The other discrepancy between our results and the UPTD predictions for our exposure situation is the assumption in the UPTD formula that 0.50 ATA O_2 is without effect regardless of time. A slight error in this number is of minimal consequence in a short term, high O_2 exposure for which the UPTD concept was originally derived. However in much longer exposures at O_2 levels only slightly above 0.50 ATA O_2, any inaccuracy in this assumed number would be greatly magnified. Clark and Lambertsen originally used 0.6 ATA as a critical level (11) and later revised this to 0.5 ATA (10). Their original prediction may be a better approximation for predicting decrements in the VC, especially when exposure times are lengthy.

Flow Rates

After corrections for gas density, the cause of the decrease in maximum flow rates is only speculative, but could include changes in lung volumes, motivational factors (pain and malaise), and neuromuscular factors. Decreases in maximum respiratory pressure and grip strength lend some support to the latter.

The delay in the onset of decreases in flow related parameters (as well as the FVC, grip, and signs and symptoms) until the late dive, decompression, and early postdive periods in AIRSAT-3 is suggestive of a toxic mechanism. It is well known that the maximum expiratory flow is greatest

near TLC and decreases as one approaches RV. Clark and Lambertsen (23) have shown that most if not all of the decrease in VC caused by POT is from the IC component and little or none from the ERV. The majority of the decrease in the peak expiratory flow rate is most likely due to the reduction in TLC causing the peak expiratory flow to be at a lesser lung volume than had occurred prior to POT. This decrement in lung volume does not appear to have as much influence on PIFR, which occurs near mid-lung position, however. These flow changes would appear to have more of a component due to neuromuscular or other changes mentioned elsewhere in this paper.

Diffusion capacity

The decrease in diffusion capacity that other studies (24-26), as well as our own, have shown, indicate that exposure to hyperoxia causes an an increase in the diffusion barrier to carbon monoxide. This may be due to 1) changes in pulmonary blood flow, 2) thickening of the alveolar-capillary membrane, 3) or ventilation-perfusion abnormalities, but we have no data to help differentiate these possibilities. We were unable to followup most subjects for more than a week, so long-term recovery data is not available.

Maximum respiratory pressure

Definite conclusions cannot be drawn from this limited data. Elevations in the CONTROL-1 subjects may be due to learning and/or a possible increase in respiratory muscle strength from multiple repetitions of this and the other pulmonary tests. The opposite effect for the AIRSAT-3 B, C, and D subjects could be due to (1) a different volume history (decreased FVC), 2) generalized fatigue and weakness, and/or 3) a neuromuscular impairment due to POT. We observed some sleeplessness, fatigue, and grip strength decreases which support the last two possibilities.

Hand grip strength

Hand grip strength was measured because it is a general indicator of overall body strength (27,28). The decrement in it would tend to support one of the possible theories for FVC decreases during POT, i.e., a neuromuscular impairment (10, 23, 29, 30, 31), although it could also be decreased because of generalized fatigue. There was no significant change for the three CONTROL-1 Dive subjects. In contrast a slight drop in the AIRSAT-3 subjects was seen.

CONCLUSIONS

a) Humans can tolerate hyperbaric air at 2.8 ATA (PO_2 =0.59 ATA or 499 torr) with brief exposures to even higher levels for periods of at least a week with only minimal signs and symptoms consistent with pulmonary oxygen toxicity.

b) Humans can tolerate 24 hours exposure to 5 ATA air (PO_2 =1.05 ATA or 795 torr) with a subsequent air decompression, and demonstrate only moderate symptoms and mild to moderate decrements in the vital capacity. Complete recovery occurred in all cases.

c) There is a large individual variation in both susceptibility to and recovery from hyperoxia.

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TABLE 1. SUBJECT PHYSICAL CHARACTERISTICS* FOR AIRSAT-1, 2, AND 3 DIVES

EXPERIMENT	n	AGE (yr)	HEIGHT (cm)	WEIGHT (KG)	BODY FAT (%)
AIRSAT-1	11	25.5±5.0	178±4.0	77.6±7.0	19.8±4.5
		(20-35)	(172-185)	(69.9-91.2)	(14.9-28.0)
AIRSAT-2	12	23.8±3.3	180±8.0	77.3±9.0	16.4±3.6
		(21-32)	(162-189)	(61.7-95.7)	(11.0-22.0)
AIRSAT-3	12	30.1±3.6	176±9.0	81.4±10.0	22.1±4.3
		(25-36)	(162-194)	(68.4-104.4	(15.0-27.0)
CONTROL-1	3	20.3±0.6	179.1±6.5	77.1±3.6	11.2±2.1
		(20-21)	(172-184)	(74.4-81.2)	(9.3-13.5)

* units expressed as mean ± standard deviation, (range)

TABLE 2. DIFFUSION CAPACITY ($D_{L_{CO, sb}}$) in mm CO/min/mm Hg for AIRSAT-1, 2, and 3 Dives

	PREDIVE CONTROL	POSTDIVE #1 (0 days)	POSTDIVE #2 (1 day)	POSTDIVE #3 (2 days)	POSTDIVE #4 (3 days)	POSTDIVE #5 (6 days)
AIRSAT-1						
$\overline{X}$	43.2	40.4				
n	5	5				
S.D.	2.93	8.12				
AIRSAT-2						
$\overline{X}$	40.3	33.8				
n	3	3				
S.D.	1.50	2.07				
AIRSAT-3						
$\overline{X}$	29.5	26.2	25.4**	24.7**	24.8*	27.6
n	6	6	6	6	6	6
S.D.	3.58	6.42	5.70	5.06	5.34	6.60

* Statistically significant at 5% level (Paired t-test)

** Statistically significant at 2% level (Paired t-test)

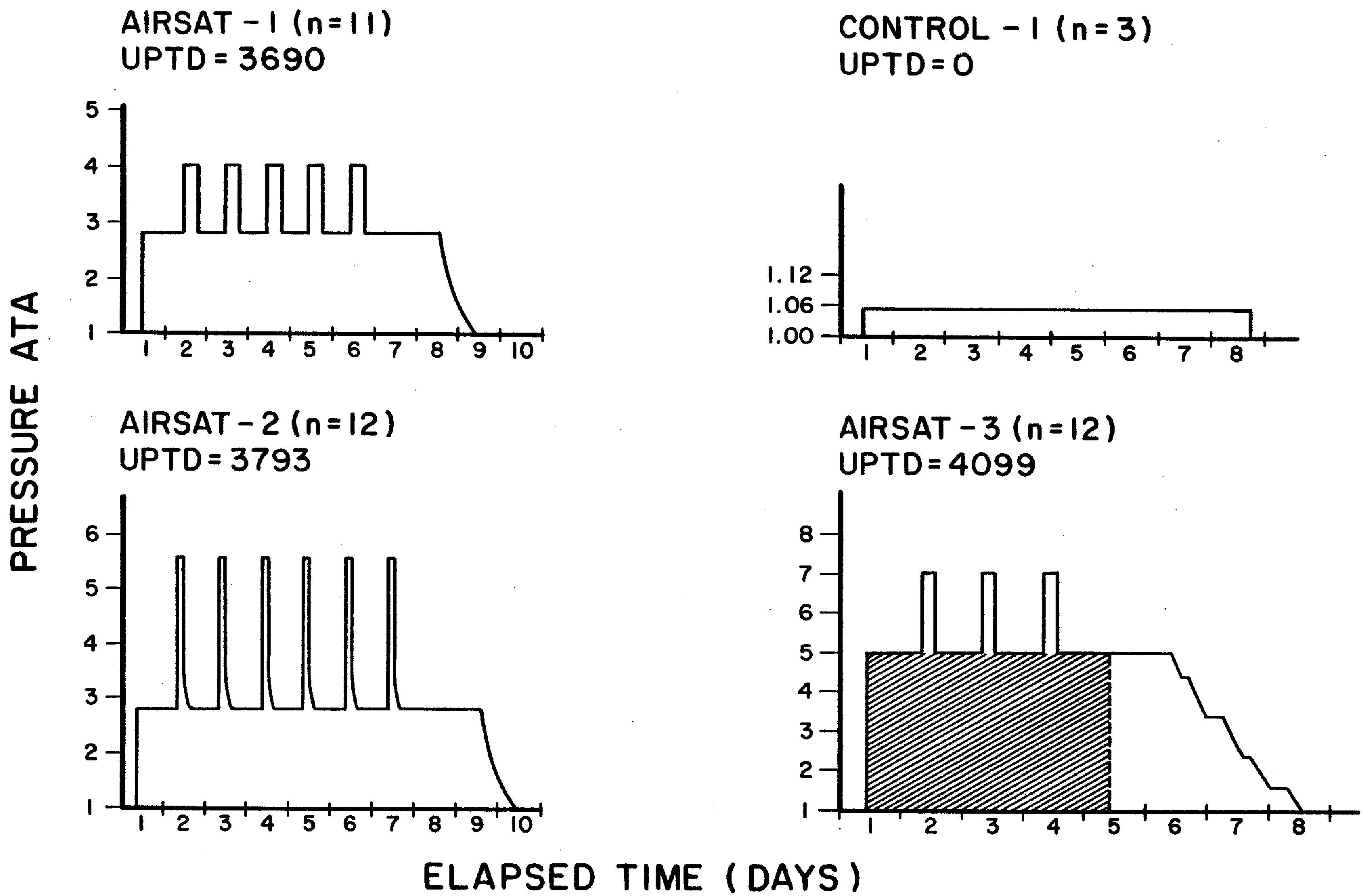

Figure 1. Pressurization profiles for the exposures. The clear areas indicate that air was the breathing media. Crosshatched areas indicate that other nitrogen-oxygen mixtures were used (oxygen partial pressure = 0.30 ATA).

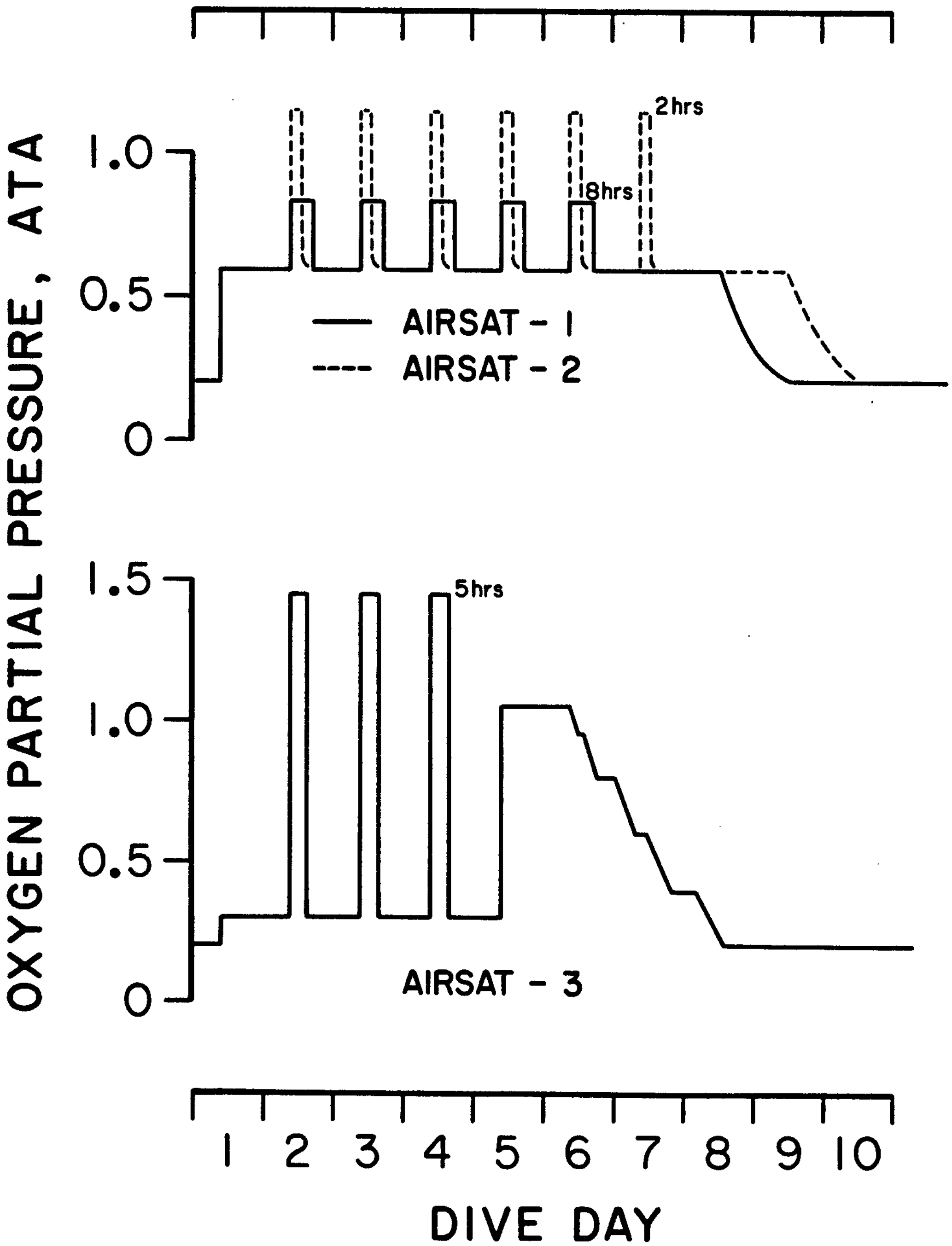

Figure 2. Oxygen partial pressure profiles for AIRSAT-1, 2,and 3. The diluent was nitrogen in all cases.

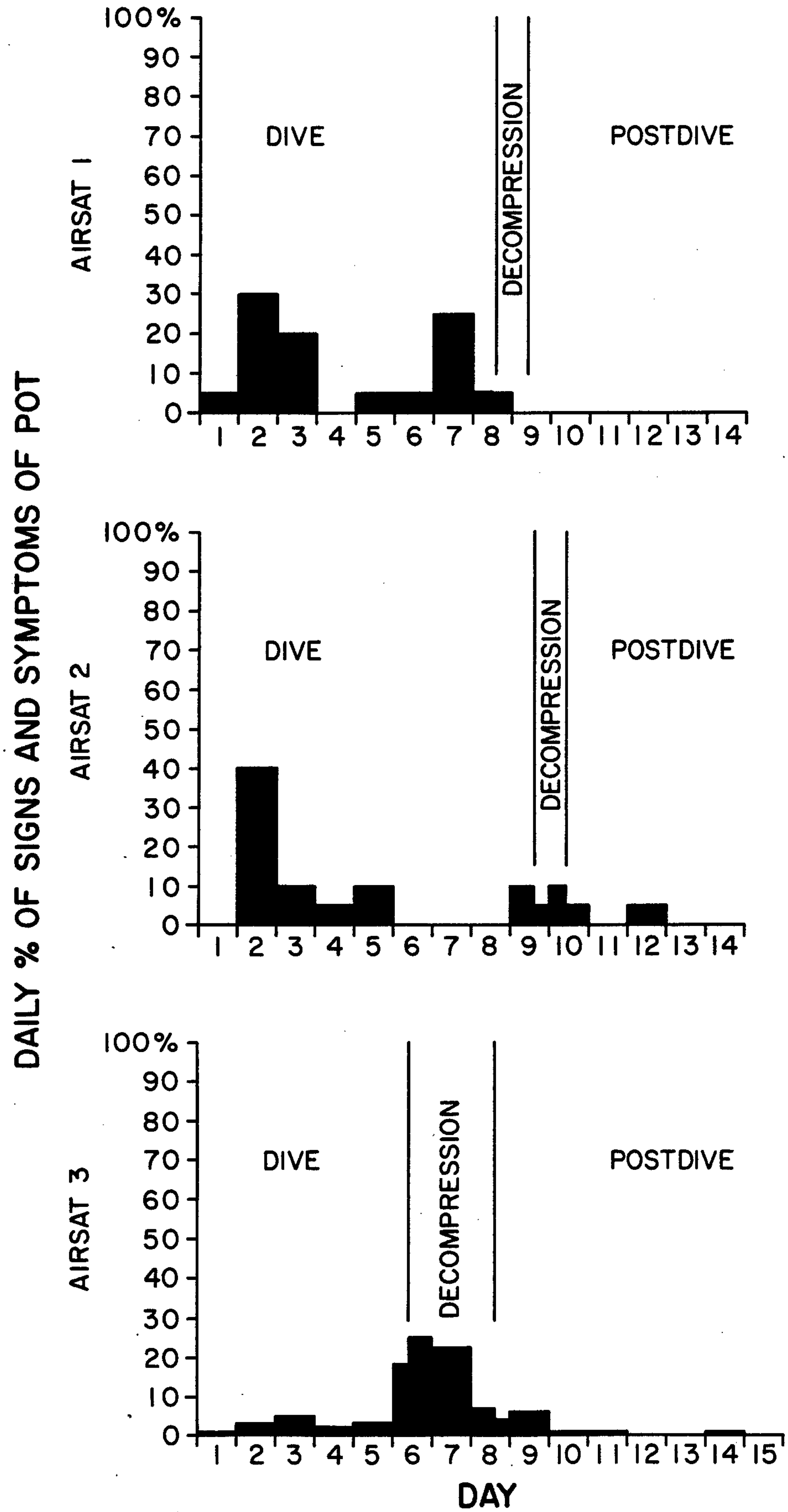

Figure 3. Time course of signs and symptoms for AIRSAT 1, 2, and 3.

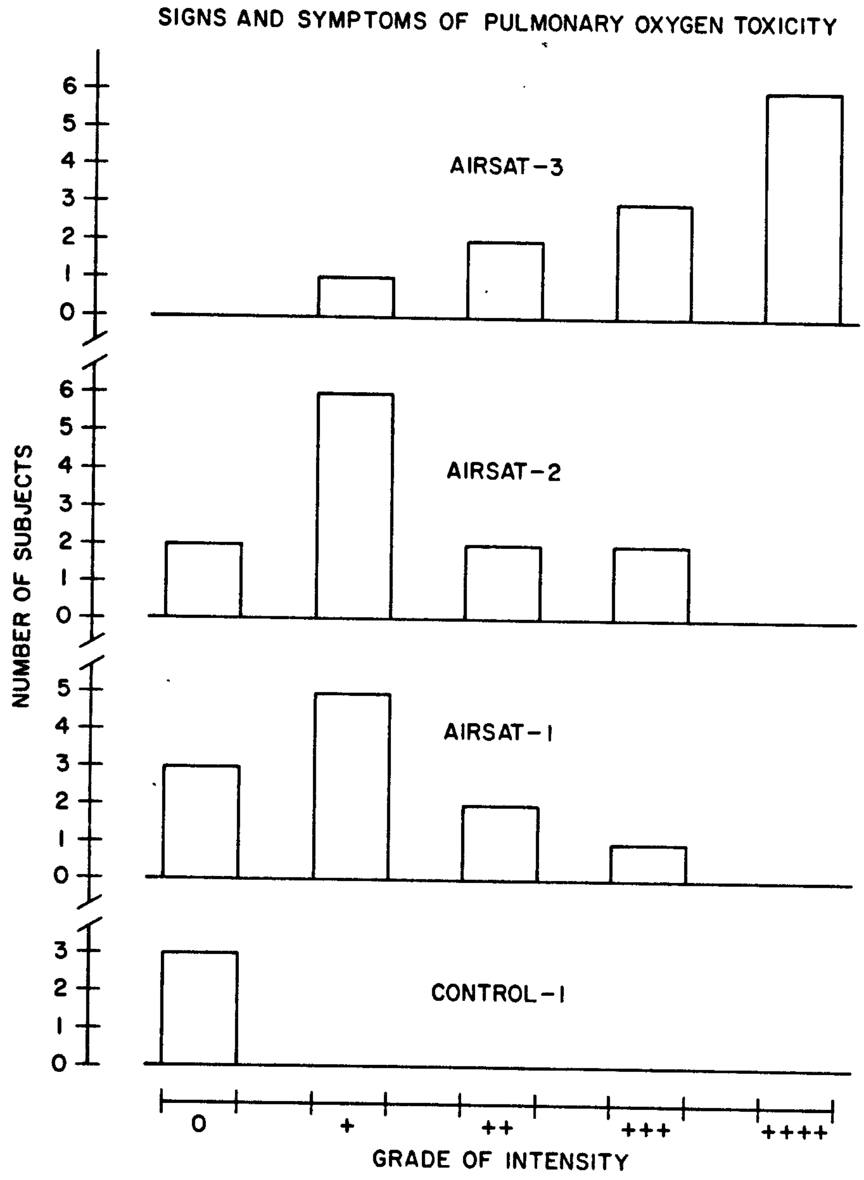

Figure 4. Number of subjects with each grade or intensity of signs and symptoms of POT (0, +, ++, +++, ++++) for CONTROL-1 and AIRSAT-1, 2, and 3.

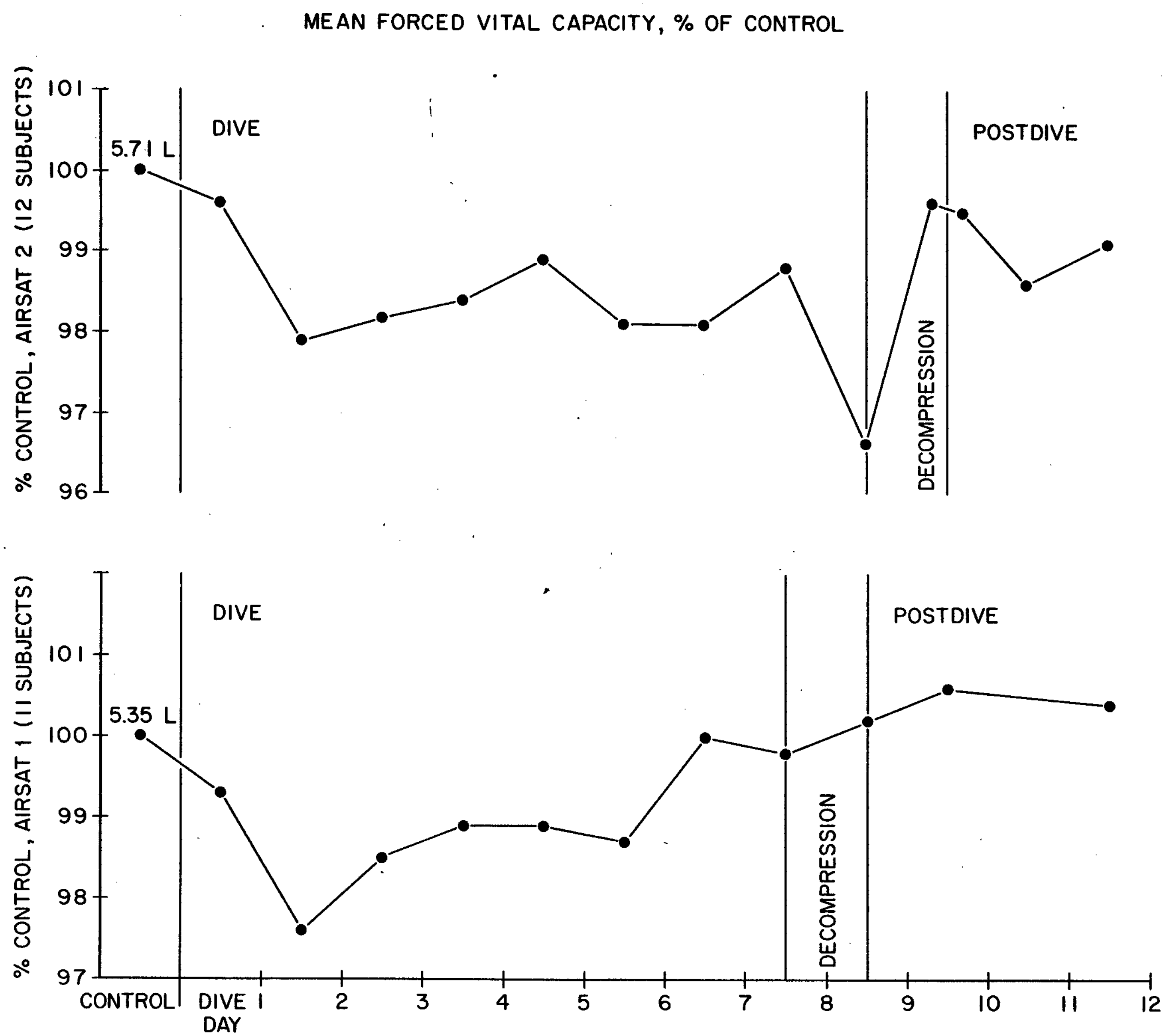

Figure 5. Mean forced vital capacity from predive control through postdive recovery periods for AIRSAT 1 and 2.

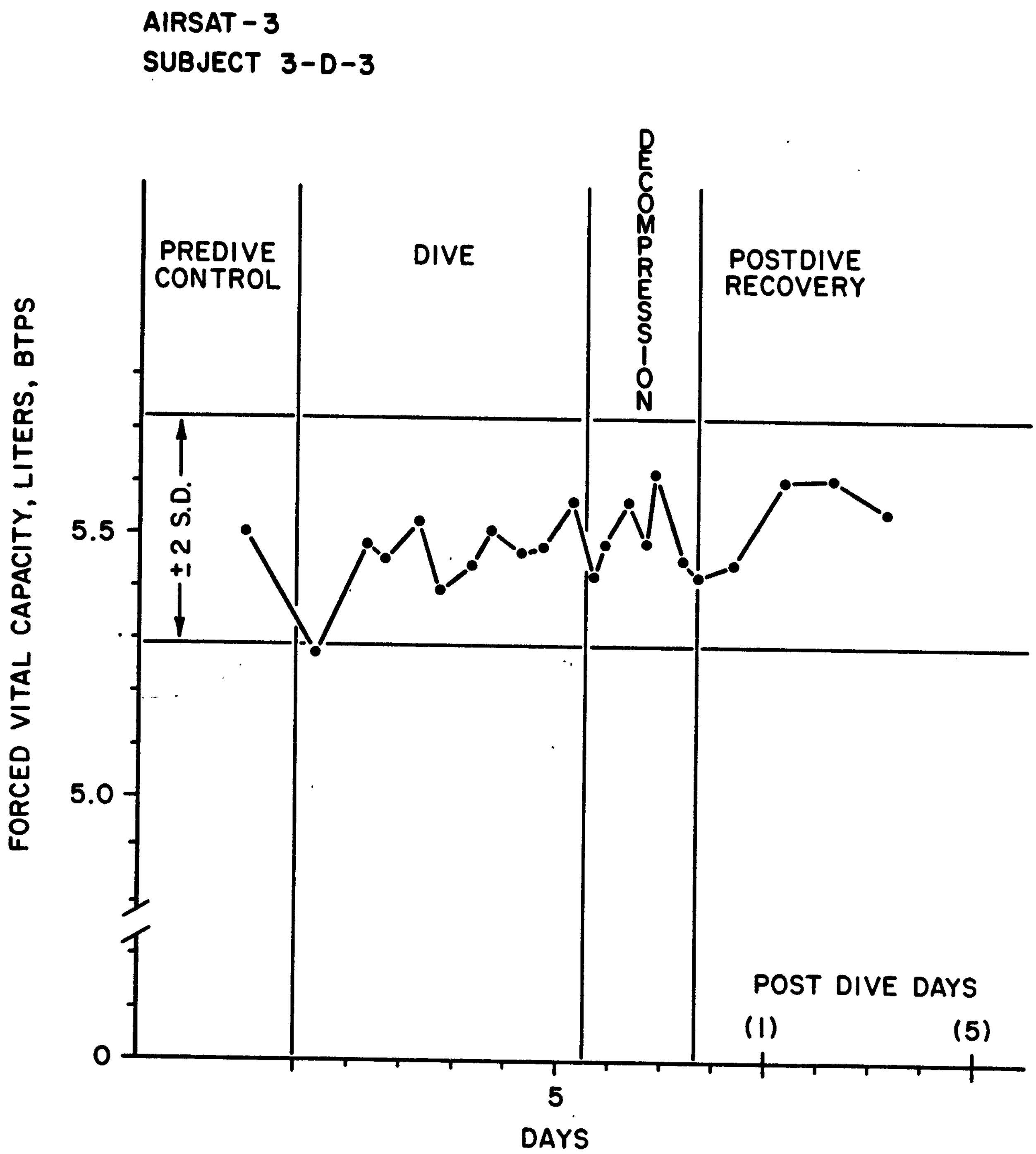

Figure 6. This shows an AIRSAT 3 subject with a minimal decrement in his FVC.

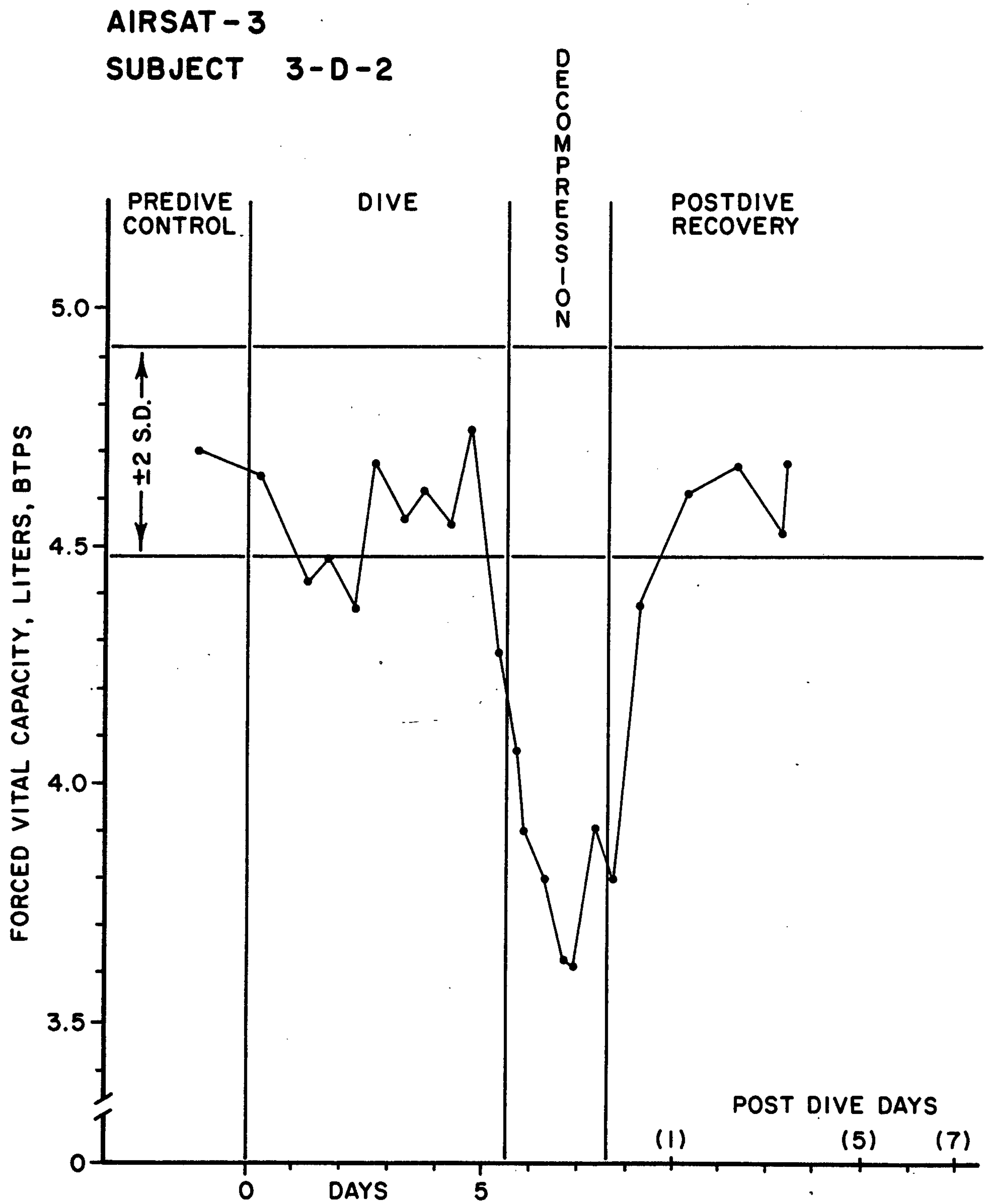

Figure 7. This shows an AIRSAT 3 subject with a large decrement in his FVC.

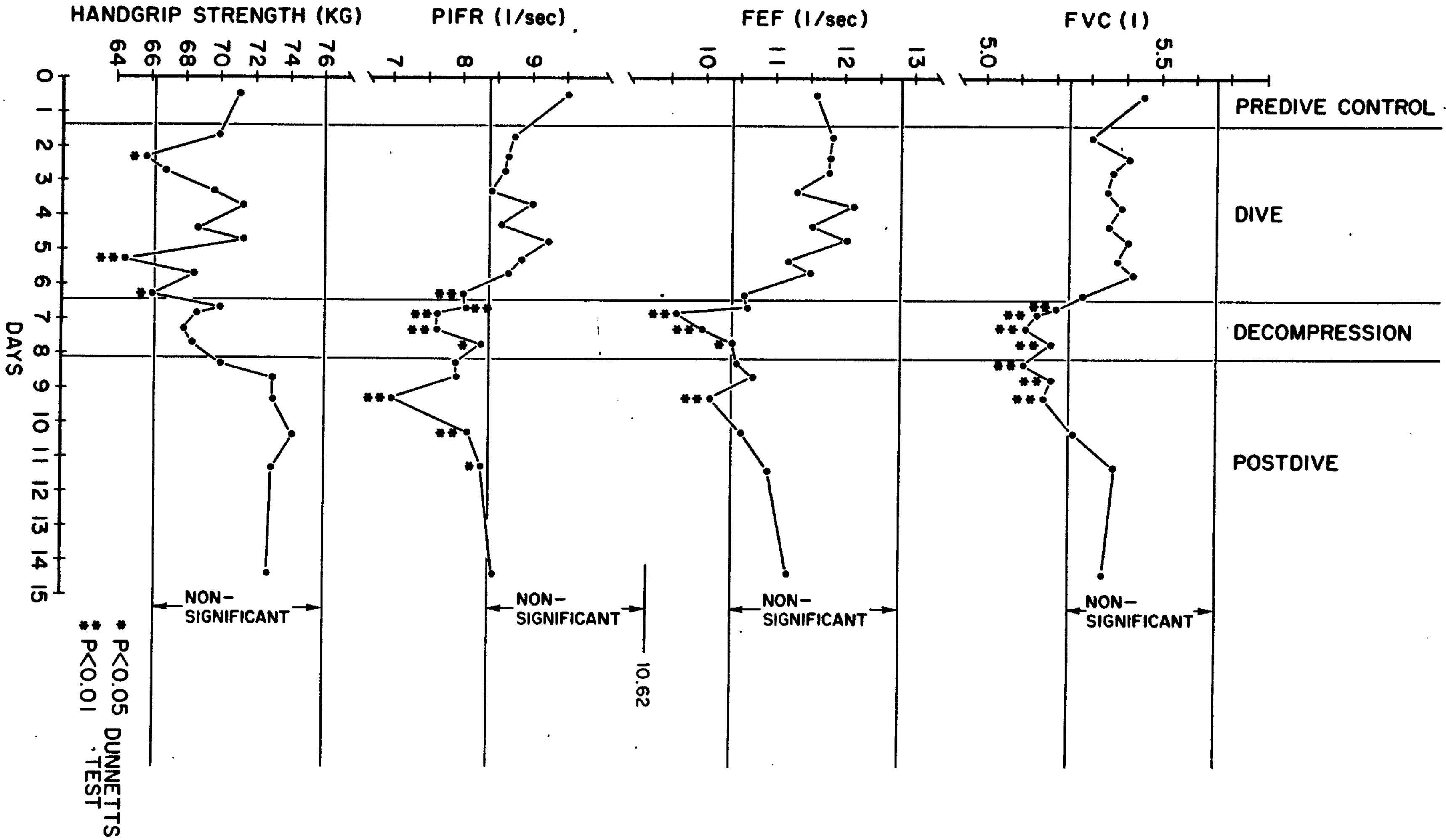

Figure 8. Mean values for FVC, FEF, PIFR, and handgrip strength at each sampling time for the 12 subjects in AIRSAT 3. Lines show the limits of no significant change by Dunnett's Test. Values outside these lines are significant at the p <0.05 (*) or p <0.01 (**) levels.

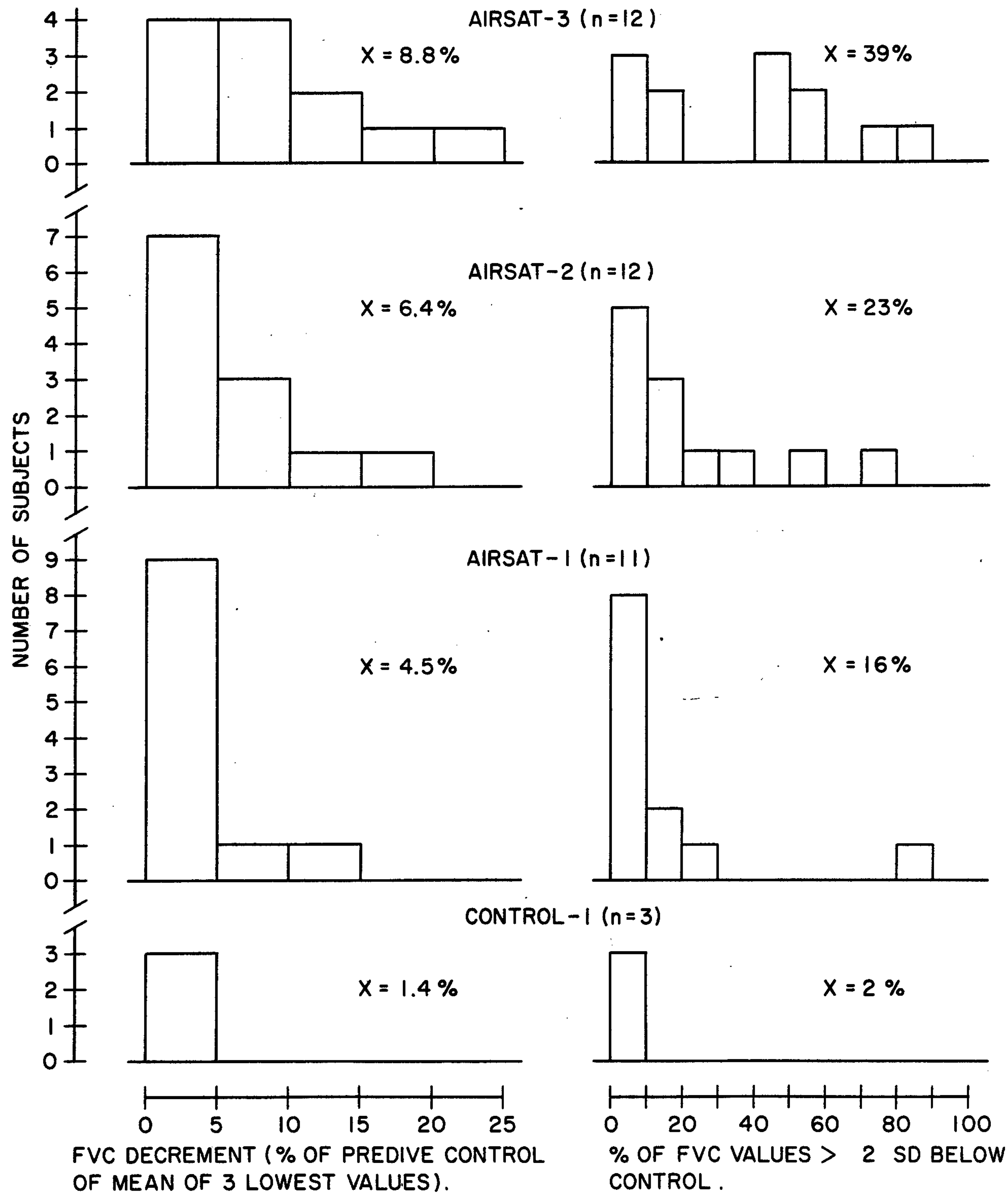

Figure 9. The left side shows the number of subjects for each exposure that the mean of the lowest 3 values of FVC decreased 0-5, 5-10%, etc. from control. The right side shows the number of subjects having 0-10, 10-20%, etc of post control FVC values >2 SD below predive control.

FOOTNOTES

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Submarine escape and rescue; Deep submergence rescue vehicle; Pulmonary oxygen toxicity; Chamber diving; Compressed air diving; Vital capacity

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

Three saturation dive profiles (11 separate dives) utilizing a total of 35 human subjects were completed. They were AIRSAT-1 (2.82 ATA air saturation, five 8-hour excursions to 4.03 ATA), AIRSAT-2 (2.82 ATA air saturation, six 2-hour excursions to 5.55 ATA), and AIRSAT-3 (0.3 ATA O_2 at 5 ATA, three 5-hour air excursions to 7 ATA). AIRSAT-3 involved a 24 hour exposure to 5 ATA air prior to decompression. Pulmonary function was measured in all subjects throughout the exposures. Signs and symptoms of

pulmonary oxygen toxicity and significant decreases in forced vital capacity and single breath diffusing capacity were observed. Marked individual variability was apparent in all exposures. Air at 2.82 ATA (60 fswg, 0.59 ATA O_2) is very close to the oxygen toxicity threshold, and air at 5.00 ATA (132 fswg, 1.05 ATA O_2) is well beyond it.